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Set Name side by side	Query	Hit Count	Set Name result set
DB=PGPB,USPT,USOC,EPAB,DWPI; PLUR=YES; OP=ADJ			
L6	L5 with l4	185	L6
L5	tat with (muta$ or delet$)	888	L5
L4	FIV or CEAV or HIV-1 or lentivi$	25144	L4
L3	6312683.pn.	2	L3
L2	6312682.pn.	2	L2
L1	6312382.pn.	2	L1

END OF SEARCH HISTORY

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#

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L6: Entry 95 of 185 File: USPT Jul 2, 2002

DOCUMENT-IDENTIFIER: US 6414132 B1
TITLE: Method of eliminating inhibitory/instability regions of mRNA

Detailed Description Text (119):
To identify elements that have a down regulatory effect in vivo, fragments of env cDNA were inserted into two different test expression vectors, p19 and p37M1-10D. These vectors contain a strong promoter for rapid detection of the gene product, such as the HIV-1 LTR in the presence of Tat, and an indicator gene that is expressed at high levels and can easily be assayed such as p19.sup.gag g of RSV or the mutated p37.sup.gag gene of HIV-1 (p37M1-10D), neither of which contains any known INS-like elements. Expression vector p19 contains the HIV-1 LTR promoter, the RSV p19.sup.gag matrix gene, and HIV-1 sequences starting at KpnI (nt 8561) including the complete 3' LTR (Schwartz, et al., J. Virol. 66:7176-7182 (1992). Upon transfection into HLtat cells high levels of p19gag are constitutively produced and are visualized on Western blots. Expression vector p37M1-10D contains the HIV-1 LTR promoter, the mutant p37gag (M1-10), and the 3' portion of the virus starting at KpnI (nt 8561). Upon transfection into HLtat cells this plasmid constitutively produces p37.sup.gag that can be quantitated by the HIV-1 p24.sup.gag antigen capture assay.

Previous Doc Next Doc Go to Doc#

L6: Entry 120 of 185 File: USPT Mar 6, 2001

DOCUMENT-IDENTIFIER: US 6197755 B1
TITLE: Compositions and methods for delivery of genetic material

Detailed Description Text (346):
The pREG plasmid is constructed in a stepwise fashion, and each intermediate can be tested for protein expression before construction is continued. An expression vector supporting the expression of tat and rev is constructed via two steps. First, an amplification product containing a 5' NheI site, the HIV-1 major splice donor site, the majority of the tat coding region, the region encoding the amino terminal region of the rev protein and an AvaII site is amplified from a synthetic template. This synthetic template is generated using the published sequences of HXB2 strain of HIV-1 obtained from the GenBank Database, and is altered to mutate the cysteine residues at positions 22 and 30 of the tat protein. These mutations have been shown to render tat non-functional (Kuppuswamy, et al. (1989) Nucleic Acids Research 17(9): 3551-3561).

Other Reference Publication (24):
Kuppuswamy et al., "Multiple Functional Domains of Tat, the Trans-Activator of HIV-1, Defined by Mutational Analysis", Nucleic Acids Res., 1989, 17, 3551-3561.

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#

Generate Collection Print

L6: Entry 121 of 185 File: USPT Dec 26, 2000

US-PAT-NO: 6165782
DOCUMENT-IDENTIFIER: US 6165782 A

TITLE: Method and means for producing high titer, safe, recombinant lentivirus vectors

DATE-ISSUED: December 26, 2000

US-CL-CURRENT: 435/320.1; 435/455, 435/456

APPL-NO: 09/ 271365 [PALM]
DATE FILED: March 18, 1999

PARENT-CASE:
This is a divisional of application Ser. No. 08,989,394 filed Dec. 12, 1997, now U.S. Pat. No. 5,994,136, the disclosure of which is incorporated herein by reference.

Previous Doc Next Doc Go to Doc#

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#
Generate Collection Print

L6: Entry 131 of 185 File: USPT Nov 30, 1999

DOCUMENT-IDENTIFIER: US 5994136 A
TITLE: Method and means for producing high titer, safe, recombinant lentivirus vectors

Detailed Description Text (36):
To provide a vector with an even more remote possibility of generating replication competent lentivirus, the instant invention provides for lentivirus packaging plasmids wherein tat sequences, a regulating protein which promotes viral expression through a transcriptional mechanism, are deleted functionally. Thus, the tat gene can be deleted, in part or in whole, or various point mutations or other mutations can be made to the tat sequence to render the gene non-functional. An artisan can practice known techniques to render the tat gene non-functional.

Detailed Description Text (56):
The high level of expression of the 5' LTR modified transfer vector RNA obtained in producer cells in the absence of a packaging construct indicates the producing vector is functional in the absence of a functional tat gene. Functional deletion of the tat gene as indicated for the packaging plasmid disclosed hereinabove would confer a higher level of biosafety to the lentiviral vector system given the number of pathogenetic activities associated with the tat protein. Thus, a lentiviral vector of significantly improved biosafety is a SIN transfer vector that has no wild-type copy of the HIV LTR either at the 5' or at the 3' end, which is used in conjunction with tat-less packaging vectors as described herein.

Previous Doc Next Doc Go to Doc#

L6: Entry 48 of 185 File: PGPB Jan 16, 2003

DOCUMENT-IDENTIFIER: US 20030013669 A1
TITLE: Method of treating HIV infection and related secondary infections thereof

Detail Description Paragraph:
[0076] The sequence specific nucleic acids include but are not limited to anti-protease sequences, retroviral promoter sequences, TAR sequences, HIV mutants of TAR decoy RNA, mutants TAR decoy RNA, negative mutants of the viral REV transactivator, synthetic promoters with the consensus sequence for binding of the transcription factor Sp1 and the TATA box, mutants of TATA box, TAT mutants wherein the mutations involving the seven cysteine residues, sense, anti-sense, missense derivative of CIS acting negative elements (CRS) present in the integrase gene and REV mutants, transdominant suppressors of REV (mutations involving amino acid 78 and 79), NEF-cDNA sequences and its mutants with or without U3 region sequence of the 3' LTR, POL reverse transcriptase gene mutants, POL viral integrase gene and its mutants, POL viral protease gene mutants, HIV-I LTR enhancer (-137 to -17) mutants, HIV LTR promoters starting at -78, HIV LTR sequences encoding a arginine fork from aa27 to aa38, HIV-I LTR sense sequences of the negative regulatory element (-340 to -185), HIV-1 LTR consensus sequences for binding of transcription factors of AP1/COUP, NFAT-1, USF, TCF-.alpha., NF-KB, TCF-1a, TBP, and inhibitors of the consensus sequence, LTR NFkB mutants (-104 to -80), LTR Sp1 (GC box) binding site and TATA box mutants, LTR GAG gene sequence mutants, LTR mutants (-454 to +180), LTR genomic repeats at +80, LTR regions responsive for cellular transcription factors between and to the left of U3 to -454 extending to -7, 3' LTR and its variants, 5' LTR and its variants, LTR variants, inhibitors of UBP-1 or LBP-1 binding sequence (-5 to +82), ENV, GAG, POL gene sequences placed 3' of the REV mutant codon, short sequence mutants (15-60 mer), and host DNA sequences of preferred targets for proviral integration.

CLAIMS:

25. The method of claim 1 wherein the sequence specific nucleic acid is selected from the group consisting of an anti-protease sequence, a retroviral promoter sequence, a TAR sequence, a HIV mutant of TAR decoy RNA, a mutant TAR decoy RNA, a negative mutant of the viral REV transactivator, a synthetic promoter with the consensus sequence for binding of the transcription factor a Sp1 and the TATA box, a mutant of TATA box, a TAT mutant wherein the mutations involving the seven cysteine residues, a sense, anti-sense, missense derivative of CIS acting negative elements (CRS) present in the integrase gene and REV mutant, a transdominant suppressor of REV (mutations involving amino acid 78 and 79), a NEF-cDNA sequence and its mutant with or without U3 region sequence of the 3LTR, a POL reverse transcriptase gene mutant, a POL viral integrase gene and its mutant, a POL viral protease gene mutant, a HIV-I LTR enhancer (-137 to -17) mutant, a HIV LTR promoter starting at -78, a HIV LTR sequence encoding a arginine fork from aa27 to aa38, a HIV-I LTR sense sequence of the negative regulatory element (-340 to -185), a HIV-1 LTR consensus sequence for binding of transcription factors of Ap1/COUP, NFAT-1, USF, TCF-.alpha., NF-KB, TCF-1a, TBP, and an inhibitor of the consensus sequence, a LTR NFKB mutant (-104 to -80), a LTR Sp1 (GC box) binding site and TATA box mutant, a LTR GAG gene sequence mutant, a LTR mutant (-454 to +180), a LTR genomic repeat at +80, a LTR region responsive for cellular transcription factors between and to the left of U3 to -454 extending to -7, a 3' LTR and its variant, a 5' LTR and its

variant, a LTR variant, an inhibitor of UBP-1 or LBP-1 binding sequence (-5 to +82), a ENV, GAG, POL gene sequences placed 3' of the REV mutant codon, a short sequence mutant (15-60 mer) and a host DNA sequence of preferred targets for proviral integration.

Previous Doc Next Doc Go to Doc#